On the cause of so-called Phosphorus Necrosis of the Jaw in Match-Workers.

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ON THE CAUSE OF SO-CALLED PHOSPHORUS NECROSIS OF THE JAW IN MATCH-WORKERS.

Historical Summary.

PHOSPHORUS was first used in the manufacture of matches in 1833 at Vienna. Shortly after its introduction cases of necrosis of the bones of the upper and lower jaws occurred among the workpeople employed in match factories. The condition was first described by Lorinser, who, between the years 1839 and 1845, saw nine cases in Vienna, and immediately after others were reported in Nürnberg, Strassburg, Berlin, Paris, Manchester, and London as having occurred among the workers in match factories. Improvements in ventilation and in manufacturing machinery have greatly diminished its frequency, but it has continued to be not uncommon, and is widely recognised as a risk incurred by those who work with phosphorus. The clinical symptoms have been fully described by Lorinser, Heyfelder, von Bibra and Geist, Harrison, Roussel, and others. In addition to their more systematic descriptions many isolated cases have been put on record by different writers, all of which agree substantially in their main features.

Symptoms and Course.

The disease may begin after a few weeks or months or many years of employment in the factory, generally with toothache, and always in a carious tooth or in the socket of one which has

been extracted. Its further progress may be very slow, the pain and inflammation remaining slight, and confined to the immediate area of the one tooth. Sometimes, however, it runs a much more acute course, and may involve a large part of the jaw in a few weeks or months. In either case, when the process once begins to extend, the gum becomes red, tense, and swollen, and the swelling may then resolve itself into a circumscribed phlegmon, or may form a large tumour which spreads to the neighbouring soft parts, causing a swollen appearance of the lips and cheeks, or the regions under the lower jaw. The abscess then bursts or is opened, when it discharges stinking pus, and in its place an ulcer quickly forms, which lays bare the bone. The discharge is greenish, greyish, or sanious, and contains a quantity of bony detritus. The teeth become loose and fall out at the diseased part; the gum becomes livid, and fistulous openings form in it from which pus discharges continuously into the mouth, or the pus may burrow and discharge externally through the skin of the face by several small openings. On probing these openings, bare bone can be felt. Later the gum disappears, and exposes the alveolar arch and other parts of the jaw, the bare bone having a brownish or dirty-grey colour; its surface is roughened and eroded, evidently in a condition of caries, and the pus can be seen oozing from its surface. The salivary and neighbouring lymph glands are hard and swollen. Larger or smaller portions of the jaw-bone then become necrosed, in process of time get loosened and detached from the rest, and ultimately exfoliate or can be easily removed with forceps. There is great difficulty in chewing and swallowing, and the patient has to live on soft food and liquids. The process may last for months or years, and may be entirely confined to the upper or lower jaw, the general health remaining fairly good. It may spread from one part of the jaw to another, or from one jaw to the other.

In the most favourable cases, after portions of the jaw have necrosed and been removed, the whole process may cease, new bone is formed from the periosteum, and cicatrisation of the soft

parts takes place. But there is always more or less deformity, sometimes very slight, sometimes very severe, and the alveolar arch is never restored.

In other cases the disease, instead of healing, spreads locally, and involves more of the bone; the patient becomes cachectic, feverish, and wasted, and ultimately dies of pulmonary phthisis, general tuberculosis, or some other tuberculous affection. A few cases run a very acute course, both as regards the local and general conditions, there being very great destruction of bone and very severe systemic disturbance, death occurring in two or three months.

Pathology.

As regards the bone, the condition is one of cario-necrosis. There is chronic ostitis and periostitis, and it differs in no respect from the same lesion as seen in other bones and from various causes.

It has long been held that this necrosis, or cario-necrosis as it should rather be termed, is due to a specific action of phosphorus fumes on the bone, these being supposed to cause a peculiar and specific kind of inflammation. If one considers, however, the whole circumstances and the clinical histories of individual cases, the conclusion must inevitably be drawn that the process is due to infection from a micro-organism. Phosphorus fumes consist chiefly of phosphorous anhydride (P_4O_6), with some phosphoric anhydride (P_2O_5), and during the oxidation either ozone or hydrogen peroxide is also formed in small amount. From what we know of suppurative processes, it is inconceivable that any of these bodies can cause a chronic purulent inflammation of bone such as has just been described as occurring in "phossy jaw."

Acting on this idea I applied to Mr Cornelius E. Garman, surgeon to Messrs Bryant & May's match factory, who very kindly has supplied me with specimens of pus from six cases of phosphorus necrosis of the jaw under his care at the present time. In every case the pus was very fœtid and was greenish,

or brownish, or greyish in colour. Attempts to make cultivations from the pus revealed the presence of staphylococcus albus, streptococci, and numerous other organisms, none of which could reasonably be regarded as the cause of the cario-necrosis.

Presence of Tubercle Bacilli.

It is well known that tubercle bacilli cannot be cultivated from pus, but on staining cover-glass preparations of the pus by the Ziehl-Neelsen method the bacillus tuberculosis was found in every case. As is usual in the discharge from tuberculous bone, the organisms were few in number and difficult to find except on the closest and most careful examination. On centrifuging the pus and then examining the lowest layer they were more easily detected. Sometimes several cover-glasses had to be examined before any of the organisms were seen. Most of the bacilli were perfectly typical in appearance, others were small and thick, resembling the forms usually found in the urine. They were scattered about singly, or in small clumps, or in groups of one or several dozens.

Inoculation of guinea-pigs with the pus did not infect these animals with tubercle, and hence the bacilli must be regarded as being either dead or as having almost entirely lost their infective virulence. It is now proved, however, that tubercle bacilli in this condition are quite capable of setting up and maintaining local suppuration and irritation for an indefinite time. Besides, they are assisted by the action of the pyogenic organisms with which the pus swarmed. The condition of the tubercle bacilli is probably to be explained by the fact that all the cases which I have had an opportunity of examining are recovering, and have been under treatment for very long periods with antiseptic mouth washes, &c.

If further proof of the tuberculous nature of the jaw disease were wanted, it is to be found in looking through the accounts of *post-mortem* examinations of fatal cases. In most death occurs from tuberculosis of the lungs. Whether this is due to infec-

tion from the jaw tubercle, or whether the phosphorus fumes damage the lungs, and make them more susceptible to direct infection, I am unable to say. General tuberculosis is also not uncommon, while tubercle of the abdominal glands and tuberculous ulcers of the intestine are almost invariable, these last arising certainly from infection by swallowing the pus. Abscess in the brain, purulent pleurisy and tubercular meningitis are also occasional causes of death. Hectic fever and emaciation always accompany fatal cases.

The condition generally is exactly similar to what is seen in tuberculosis of the jaw in cattle, and in tuberculous disease of other bones in man.

The presence of the tubercle bacillus can hardly be regarded as fortuitous, seeing that it was found in every case, and its presence is held, so far as our present knowledge goes at least, to be proof positive of the tuberculous origin of any lesion.

Action of Phosphorus.

The part which the phosphorus plays in the process is not far to seek. The acid fumes (phosphorous and phosphoric acids) produced by its oxidation in the air have no effect on bone covered by gum or mucuous membrane; but when they can penetrate to the bone directly through the aperture formed by a decayed or extracted tooth or by any injury, they erode the bone, weaken its nutrition and resisting power at this small spot, and make it susceptible to infection by tubercle bacilli. The bacilli having made good their foothold, spread slowly in some cases and with disastrous rapidity in others. I think I am correct in saying that the great majority of workers in match factories have carious teeth, and yet only a very small proportion of them become affected with cario-necrosis of the jaw—namely, those of them who, owing to their home surroundings or to individual predisposition, become readily infected by the tubercle bacillus. Von Bibra and Geist state that the disease may occur weeks or months after the patient has left

the match factory, and in one of their reported cases the woman had actually been eighteen months away from the work before any symptoms began. This in itself is almost complete proof that the phosphorus fumes are only a predisposing cause, and that the disease depends on subsequent infection.

It is well known that von Bibra and Geist, and later Wegner, produced suppuration and cario-necrosis in the jaws of rabbits by injuring the periosteum and then exposing the animals to phosphorus fumes (on uninjured rabbits the fumes had no effect). The rabbits all died in from five to ten weeks' time, and were found to have tubercle of the lungs. I experimented in a different way, as it is evident that these animals had become rapidly infected from the laboratory cages in which they were kept. I got new wooden hutches made, placed them in a room where animals had not been previously kept, and kept them scrupulously clean. In the hutches pieces of phosphorus were placed on a mortar on damp earth (to avoid risk of fire) in such quantity that the cages were constantly filled with the fumes in much greater amount than can possibly occur in any factory. Four rabbits were then placed in the hutches after the periosteum and gum had been removed over a considerable portion of the upper and lower jaws in each. In one a tooth was loosened in addition, the operations being all performed under chloroform. They seemed to suffer no inconvenience either from the operation or from living in the phosphorus-fume atmosphere. It has been very difficult to prevent the gum growing over the exposed bone, and after many weeks there is not the slightest trace of any jaw affection. The exposed surface of bone has become slightly eroded and rough, but whether from the action of the acid fumes or from that of the bacilli of the mouth it is impossible to decide.

Treatment.

The treatment hitherto pursued in cases of phosphorus jaw has been to wash out the mouth with deodorant and antiseptic

lotions, and wait until the necrosed pieces of bone come away. This is always extremely tedious, and may last many years. In extreme cases the whole lower jaw, or half of it, or parts of the upper jaw have been excised. Sometimes by so doing the whole of the infected portion may be removed, but frequently the disease has again broken out in a neighbouring part of the bone. It is evident, however, that early operative interference is called for, and that the original tuberculous focus at the root of the tooth should be removed at once.

Prophylaxis.

As regards prophylaxis, there is absolutely no risk so long as the bone remains protected by gum, and even when carious teeth are present the entrance of the bacilli can be prevented by careful stopping. Efficient ventilation of the workshops will dilute the acid fumes arising from the phosphorus, and make them less active in injuring exposed bone.

The infection with the tubercle bacilli is a matter quite apart from the factories, and cannot be controlled either by State regulations or workshop rules. It is acquired—as other tuberculous affections are acquired—by certain persons and not by others, and owing to the present all-pervading frequency of the organism persons with exposed bone eroded by acid fumes, and living under bad hygienic conditions, are apt to become infected. Whether the fumes also weaken the mucous membrane of the lung alveoli and predispose to pulmonary phthisis among persons employed in match factories, I have no information which will enable me to decide. It is just possible that actinomyces or other organisms may also occasionally lodge in the weakened bone, and lead to caries and necrosis, but in those cases which I have hitherto examined I have only found the tubercle bacillus.

My great difficulty all along has been to procure a sufficient amount of clinical material to enable me to make my observa-

tions more extensive and precise, and I shall be greatly indebted if any surgeon who has cases under his care, and more especially recent ones, will supply me with specimens of the discharge.

In conclusion, I have to express my great indebtedness to Mr Garman and his son, Mr C. B. Garman, for a great deal of information and assistance, as without their active help and co-operation I could not have made these observations.